THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey
BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING
AGRICULTURAL RESEARCH ADMINISTRATION
UNITED STATES DEPARTMENT OF AGRICULTURE

Volume 33 Number 6

June 15, 1949

The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

THE PLANT DISEASE REPORTER

Issued by

THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station — Beltsville, Maryland
Volume 33 — June 15, 1949 — Number 6

IN THIS ISSUE

A severe outbreak of tobacco blue mold in seedbeds in the Carolinas and in the field in Florida, reduced prevalence of late blight this year, and local severity of cucurbit downy mildew in Florida, are features of the progress report on the Warning Service for the first four months of the year, by Paul R. Miller, page 238.

Varietal reaction in bean to the Colorado red-node virus is reported by W. D. Thomas, Jr., page 244.

A tomato variety elsewhere reported to be resistant to the curly top virus was not found to be so in Idaho, according to R. D. Watson and J. E. Kraus, page 247.

G. B. Ramsey and M. A. Smith, page 248, report heavy loss caused by the Rhizoctonia crater rot in Illinois-grown carrots in 1948; only previous records of occurrence are in New York.

T. E. Smith and S. J. Hassell report on resistance of watermelon varieties to Fusarium wilt, page 249.

A. H. Eddins and Stanley M. Burrell describe a severe outbreak of cabbage yellows in the Florida Hastings area, page 249; the disease had not been noticed in the section previously.

E. B. Lambert, G. Steiner, and Charles Drechsler, page 252, report that discovery of a nematode causing a disease of mushrooms was due to the presence of a nematode-predaceous fungus which was formerly thought to be the cause of the disease itself.

Glen Staten and Philip J. Leyendecker have found Fusarium solani to be the cause of an alfalfa root rot in New Mexico, page 254.

S. B. Fenne et al. report results of a survey for diseases of alfalfa and clover in Virginia, page 255.

Incidence of rice diseases in Arkansas in 1948 is reported by E. M. Cralley and C. Roy Adair, page 257.

Roderick Sprague and Jack P. Meiners continue their records of new diseases of Gramineae in the Western part of the country, page 259.

The wheat mosaic outbreak in Western Kansas is reported in a press release, page 271.

Consequence of the failure of the azalea flower blight organism to produce sclerotia last year in Louisiana is markedly reduced incidence of the disease this year, as reported in a follow-up note by A. G. Plakidas, page 272.

William W. Diehl reports the finding, apparently for the second time in America, of a Cladosporium scab on Buddleia, in Virginia, page 274.

Canadian black currants show blister rust immunity under field conditions in Connecticut, according to G. G. Hahn and Alton Miller, page 275.

April weather, page 277.

TOBACCO BLUE MOLD IN 1949 PROVIDES LESSON IN CONTROL: STATUS OF THE WARNING SERVICE TO APRIL 26th.

Paul R. Miller

Incidence of blue mold up to the time of this summary has provided a double object lesson. First, it has demonstrated again the certainty of control by recommended materials used in recommended fashion. Second, it has given clear proof of the danger of allowing last year's occurrence to influence attitudes toward necessity and thoroughness of control measures this year, as manifested in widespread neglect of control, delay in starting it, or improper use of control chemicals. This is brought out in the detailed report below.

Of the other diseases included in the Warning Service, late blight, on the whole, has so far not been especially serious although reports at the time of writing indicate that it is just now assuming greater importance.

Cucurbit downy mildew has not yet appeared outside of Florida where it is causing some trouble.

TOBACCO BLUE MOLD (Peronospora tabacina)

Blue mold is "raging" in the worst attack in ten years in North and South Carolina, and, if weather continues to favor its activity there and northward, 1949 will be long remembered for this disease. Several circumstances contribute to this epidemic, first among which is its very early appearance (Figure 1 and Table 1). Probably of equal importance are the favorable weather conditions and the indifference of growers to control measures because of the comparatively slight occurrence last year, which, according to T. W. Graham, "... apparently gave growers a false sense of security." He goes on with a further statement that seems to be as true of other sections as of South Carolina: "Growers in this area know from previous experience that only a sustained treating program is effective under epidemic conditions, yet few have carried out such a program." It seems to me that the best efforts of extension pathologists are required year by year to keep the farmers on an even keel with regard to the control of blue mold. Tobacco growers seem to shift from a state of excitement to one of lethargy, depending upon the severity of the disease the preceding season.

Not only indifference, but also misconception, affects grower attitude toward control. Perhaps this year will have an educational effect. As pointed out by various keymen, growers possess wholly inadequate ideas concerning both the factors involved in infection and spread of the disease and the actual use of control materials. As examples of the first category, growers in sections where plants advance too rapidly before

Table 1. Dates of first appearances of Tobacco Downy Mildew in the plant bed, 1931-1949.

State	1931	1932	1933	1934	1935	1936	1937
Fla.	May 8	Feb. 23	Feb. 20			Mar. 30	Feb. 12
Ga.	Apr. 12	Jan. 21	Feb. 20		Mar. 7	Feb.	Jan. 25
S. C.		Mar. 30	Mar. 21	Apr. 3	Apr.		Feb. 25
N. C.	Apr. 27	Feb. 23	Mar. 21	Mar. 15	Apr. 1		Mar. 10
Va.	May 29	June 8	Apr. 24		May 13		Apr. 20
Tenn.			May 5		May 3		Apr. 30
Md.	May	May 31	May 18	May 29		May 11	May 10
Ky.						June 8	May 13
Pa.		June 15	May 14				
Conn.							May 25
Mass.							June 10*
Ind.							June 13
La.	Mar. 20*						
Ohio							
Wisc.							
Canada							

State	1938	1939	1940	1941	1942	1943	1944
Fla.	Feb. 7	Feb. 9-10					
Ga.	Feb. 2	Feb. 4		Feb. 10		Early Feb.	Feb. 23*
S. C.	Mar. 14			Apr. 19			
N. C.	Mar. 7-8	Mar. 8					
Va.	Apr. 29	Apr. 10		May 5*	May 6	Apr. 27	Apr. 24
Tenn.	Apr. 19						May 12*
Md.	Apr. 21	May 1			May 4		May 4
Ky.	Apr. 29	May 7	May 15				
Pa.			May 12*				
Conn.			May 23				
Mass.	May 14	May 22	May 23	May 16	May 5-7	May 24-28	
Ind.							
La.							
Ohio		May 23					
Wisc.							
Canada	June 7+						

State	1945	1946	1947	1948	1949	Earliest appearance in 18 years
Fla.			Jan. 30	Feb. 23	Feb. 11	Jan. 30-1947
Ga.	Feb. 18		Jan. 28	Feb. 6	Jan. 11	Jan. 11-1949
S. C.	Mar. 14*	Mar. 10*	Mar. 10	Mar. 15	Feb. 26	Feb. 25-1937
N. C.			Mar. 3	Mar. 23	Mar. 4	Feb. 23-1932
Va.		Apr. 3		Apr. 4-5	Mar. 29	Mar. 29-1949
Tenn.			May 3	Apr. 30	Apr. 26	Apr. 19-1938
Md.		Apr. 12	May 5	Apr. 23	Apr. 16	Apr. 12-1946
Ky.	Apr. 28	Apr. 17	May 8-10	Apr. 29		Apr. 17-1946
Pa.	May 22-26		May 20*	May 17-21		May 12*-1940
Conn.		May 1	June 3	May 17-21		May 1-1946
Mass.			May 16	May 17-21		May 5-7-1942
Ind.						
La.						
Ohio	June 1					May 23-1939
Wisc.			June 19°+			June 19-1947
Canada			May 11	May 17-21		May 11-1947

* Approximate date. + First appearance ° Field plants

setting time frequently attempt to inoculate the beds or deliberately withhold control to keep the plants back, not understanding, or at least ignoring, the risk they run of losing their plants if conditions should suddenly become favorable for the disease. Seeding beds a month or so ahead of the normal time is sometimes resorted to, "... in the unfounded hope", as J. G. Gaines puts it, "that early plants might escape severe disease damage", but, on the contrary, this practice "almost invariably results in unseasonal, early, and severe mold at a time when the disease is much more destructive than later in the season". Also, of course, it results in an ideal situation for build-up of inoculum and spread of infection.

Possibly fitting, in its various aspects, into both categories is the contention of North Carolina growers that Fermate makes plants "too tender". Probably the main explanation for the tender plants, according to H. R. Garriss, at least in the area where this opinion is most strongly expressed, is an unnecessarily high rate of seeding, which, with protection from blue mold, results in very thick stands. Also, in many cases overfertilization is a factor. It is believed that since effective control measures for blue mold are now available, growers who plant too thickly or too early would benefit by adopting dates and rates of seeding followed before 1932.

H. R. Garriss reports various other grower opinions about control that are probably generally typical. Control has been excellent in North Carolina this year when Dithane Z-78 or Fermate were used according to recommendations. Nevertheless, some growers who did not decide to treat their beds until after blue mold appeared in them are convinced that the recommended materials are inefficient because they did not "check" the disease as growers thought they should. "This thought exists", continues this reporter, "in spite of all the teaching that materials are primarily preventatives. It is my opinion that with our present-day spray and dust treatments, any mention of curative or 'checking' qualities is dangerous propaganda for the tobacco grower to have access to. Such information is conducive to starting treatments after blue mold makes its appearance". He concludes that although considerable benefit often results from starting treatment after the disease appears, "it is a risky practice to suggest".

The addition of injurious substances to Fermate caused trouble in Georgia, reported by J. G. Gaines as follows: "Considerable and widespread commercial damage to tobacco plants has been reported in some localities from the use of Fermate dust made up with diluents other than Talc or Pyrophyllite, or where undisclosed clay stickers were used in mixing the dust. A preliminary test with one brand of this dust resulted in plant damage ranging from severe leaf burning to 80 percent plant kill from two heavy applications such as growers often use. Normal applications caused definite leaf injury. On some farms this dust damage has exceeded blue mold damage."

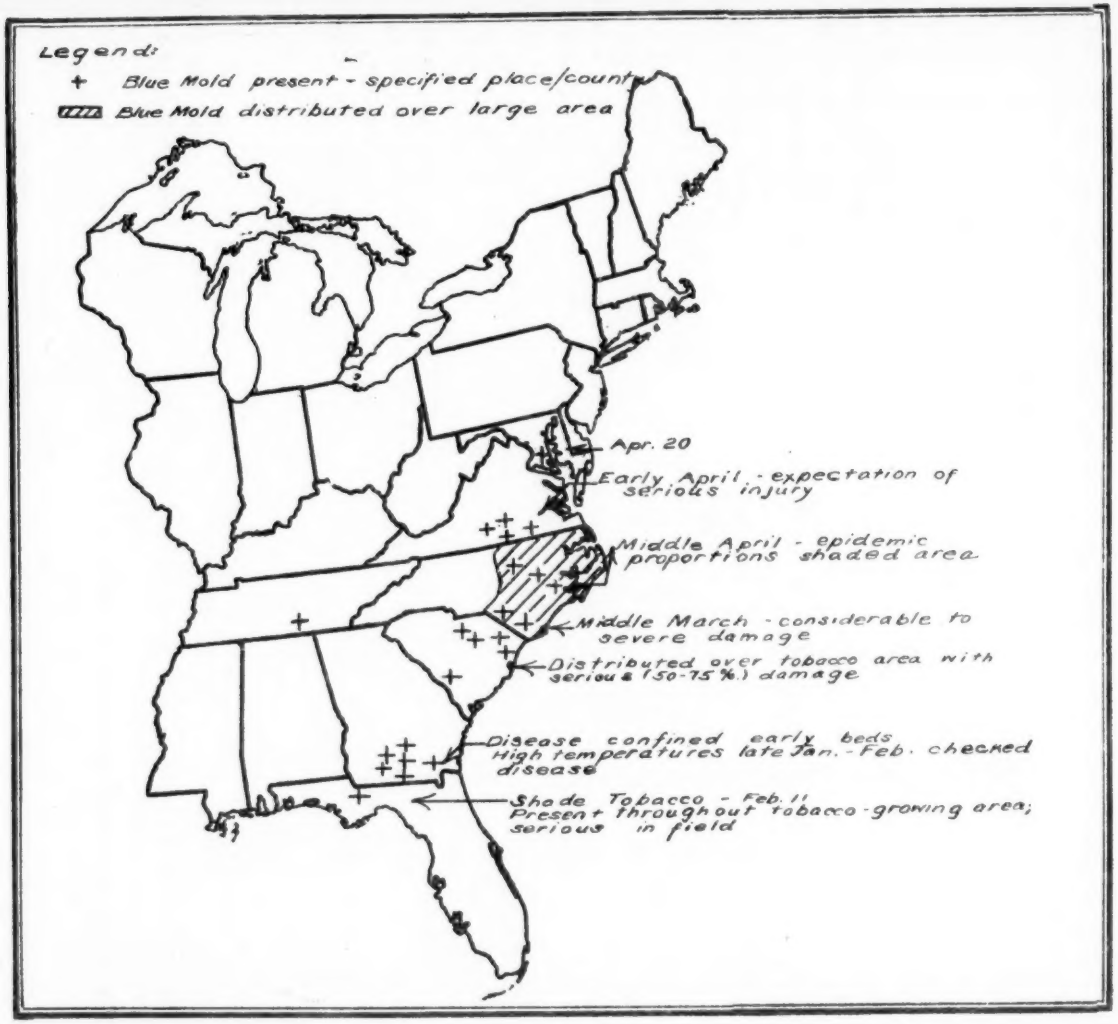

Legend:
+ Blue Mold present - specified place/county
Blue Mold distributed over large area
Apr. 20
Early April - expectation of serious injury
Middle April - epidemic proportions shaded area
Middle March - considerable to severe damage
Distributed over tobacco area with serious (50-75%) damage
Disease confined early beds High temperatures late Jan. - Feb. checked disease
Shade Tobacco - Feb. 11 Present throughout tobacco-growing area; serious in field

Fig. 1. Distribution and Severity of Tobacco Blue Mold Warning Letters Nos. 1-15 incl. - January 19-April 26, 1949.

Fig. 2. Distribution and Severity of Potato and Tomato Late Blight Warning Letters Nos. 1-15 incl. - January 19 - April 26, 1949.

A new product called "Germide", active ingredient stated to be bismuth calcium oxide 70%, "made its appearance and disappearance" in North Carolina. Garriss reports that it was "claimed to be effective as a preventive or cure when used as a dust or spray according to the manufacturer's directions. [A newspaper] article stated that Germide had been widely acclaimed in Georgia and Florida where successful experiments had been conducted in the tobacco-growing areas. Contacts with pathologists in these areas failed to reveal any knowledge of this product. Contacts with the manufacturers have failed to produce data to justify the claims made. Thanks to our sound fungicide law in North Carolina, this product has been removed from the market because of improper labelling and will not be registered for sale unless the product proves to be of merit through adequate testing".

Itinerary of the disease so far this year is easily followed from the map (Figure 1). It will be interesting to watch its further progress and compare time of appearance with dates listed in Table 1 for other years.

In GEORGIA, because of the very early appearance and the exceptional number of early-seeded beds, maximum damage was expected but did not materialize. The disease did become widespread in Georgia tobacco areas, but temperatures were too high for severe damage in late January and throughout February. Seed-bed activity in FLORIDA also was not severe.

The situation in NORTH and SOUTH CAROLINA, however, has been quite different and expectations of a severe epidemic have been fully realized, as already stated. Plants had to be brought in from outside the State to supply many South Carolina communities, while delayed planting and uneven crops were in prospect for many growers. In North Carolina, up to the middle of April, it was not yet certain whether a general plant shortage would result, but Garriss reports a shortage on some farms in the eastern part of the State, which "means movement of plants from one locality to another, a practice which we strongly discourage because of the danger of introducing the black shank and Granville wilt organisms into new locations".

Up to the end of April indications in VIRGINIA, MARYLAND, and TENNESSEE were for development similar to that in the Carolinas. Perhaps farmers in these areas may take warning from the experience of Carolina growers and become more interested in controlling the disease.

Field Outbreak in Florida. W. B. Tisdale reports that during April weather conditions were favorable for blue mold. "As a result, the most severe epidemic of blue mold has developed in the fields of flue-cured tobacco [north-central Florida] that has ever occurred. Specimens brought in from some fields have shown from four to seven of the lowest leaves completely ruined. Furthermore, many fields are showing flower 'buttons' so that more than half the crop is ruined. Since there are very few plants in the beds that are suitable for resetting, some farmers are topping the plants in the field low to encourage a sucker crop."

He states that the disease is reported to be causing field damage in the Gadsden County cigar-wrapper area also.

Downy Mildew on Other Hosts in North Carolina. Garriss reports that "On April 5, L. W. Nielsen collected seedlings of eggplant and tomato affected with a downy mildew from a plant bed near Rowland (Robeson County). The plant bed containing eggplant, pepper, and tomato seedlings was located about 300 yards from a tobacco bed in which blue mold was active. Examinations made by D. E. Ellis revealed sporulation of a Peronospora, presumably P. tabacina, on affected plants. Ellis and I visited this plant bed on April 13 and found the disease on pepper, as well as on eggplant and tomato. Sporulation was light to moderate on eggplant and very light on pepper and tomato. While the disease had caused moderate damage to eggplant and pepper, injury was negligible on tomatoes."

LATE BLIGHT (Phytophthora infestans)

Appearance and distribution of late blight on potato and tomato are shown on the map (Figure 2). Available reports so far do not indicate serious damage.

In Florida, decreased prevalence, as compared with the last few years, was manifested by easier control and moderate to slight loss. An outbreak of blight on the fall potato crop in the Belle Glade area spread rapidly in inadequately treated fields and caused heavy losses in some, but proper use of either Parzate Liquid or Dithane D-14 gave excellent control. The uniformity of blight appearance in this region suggested spore showers from outside sources as the origin of infection. In the Homestead area the slight loss to both potato and tomato crops was attributed to three factors: first, and most important, was the warmer drier weather that prevailed this season; second was the proper, frequent, and general use of fungicides; and third was the destruction of tomato seedbeds when no longer in use, thus destroying one source of infection.

Late blight was reported on potato in the Lower Rio Grande Valley of Texas, and in potato-growing counties of southern Louisiana and southern Alabama. Extent of damage has not been reported although the disease was said to spread rapidly and to have become generally distributed in the latter two sections. Very little control was attempted in Alabama, according to reports.

Except for the reports from Florida and one from Indiana, all the records of occurrence on tomato so far involve seedling transplants shipped from outside sections. Source of the infected transplants was Florida or the Texas Lower Rio Grande Valley, shown on the map.

Very careful and thorough inspection of the Georgia plant-growing area has not revealed any late blight infection of tomato seedlings grown there.

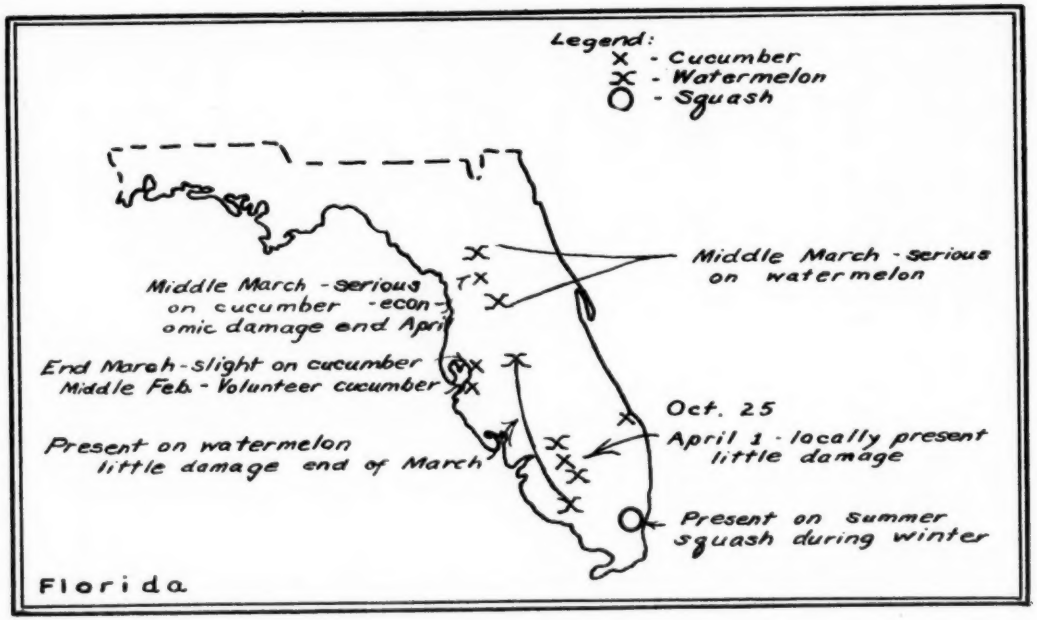

Legend:
X - Cucumber
X - Watermelon
O - Squash
Middle March - Serious on watermelon
Middle March - Serious on cucumber - economic damage end April
End March - slight on cucumber
Middle Feb. - Volunteer cucumber
Present on watermelon little damage end of March
Oct. 25
April 1 - locally present little damage
Present on summer squash during winter
Florida

Fig. 3. - Distribution and Severity of Cucurbit Downy Mildew - Warning Letters Nos. 1-15 incl. - January 19 - April 26, 1949.

In Indiana, the infection observed on tomato was in a greenhouse that presented particularly favorable conditions for late blight development. Infection apparently originated from remnants of a severely attacked fall crop. Flats of seedlings intended for the local home garden trade were affected, as well as other plants in the greenhouse.

DOWNY MILDEW (Pseudoperonospora cubensis) OF CUCURBIT CROPS

In southern Florida downy mildew has been present since the latter part of October. In the Homestead area Yellow Crookneck summer squash planted throughout the winter is commonly affected, and, since downy mildew attacks the maturing crop, no control is practiced. The disease was reported in March as very serious in central Florida on cucumbers. Dry weather later in the month reduced attack on both cucumbers and watermelon in various parts of the State, but activity apparently was reviving with more favorable conditions in the early part of April.

Dithane D-14, Parzate, Zerlate, various copper compounds, and sulfur were reported as being used for control. In specific instances mentioned, Zerlate and Dithane D-14 gave excellent control. In some cases, however, Dithane D-14 and Parzate were not as effective as expected.

Figure 3 shows distribution and severity on the various hosts.

DIVISON OF MYCOLOGY AND DISEASE SURVEY

REACTION OF DIFFERENT BEAN VARIETIES TO THE COLORADO RED-NODE VIRUS[1]

W. D. Thomas, Jr.[2]

There has been an increasing prevalence of a disease on field beans in Colorado since 1946 resembling that described by Virgin (3) in 1943. Some of the symptoms expressed and varietal reactions obtained, have resembled those of shiny pod (4,5) and others of a strain of bean virus 2 (1). However, because of some symptomological differences (2), the disease tentatively has been designated as Colorado red node. In an effort to identify the Colorado red node virus and to determine its possible relation to other similar viroses on field beans, the reaction of different bean varieties to the virus was studied.

Fresh sap, obtained from crushed infected leaves, was rubbed onto carborundum-coated leaves of 17 bean varieties in the greenhouse (Table 1). Five replicates of 5 plants each were used for each variety. Plants, from which the inoculum was obtained, manifested a red necrosis of the veins and sunken red lesions along the stems and at nodes. No mottle was evident. Typical symptoms of red node were expressed, by artificially inoculated plants, within an incubation period of 10 days at 80°F.

In Table 1 are recorded the average number of plants that expressed symptoms, and the types of symptoms produced. No infection was evident on Kentucky Wonder White No. 780 or Kentucky Wonder Brown No. 814. Leaf symptoms of red node were expressed on all other varieties, regardless of the presence or absence of stem symptoms. No red-node stem symptoms were obtained on US No. 5 Refugee, California Stringless Wax or Pinto No. 650. The top necrosis on Great Northern UI 15, Michelite, and Bountiful is in accordance with Grogan's observations (1).

Test plants were allowed to mature. Mosaic-resistant Idaho selections occasionally produced pods that exhibited symptoms of shiny pod and Colorado red node simultaneously. This may indicate that either certain environmental conditions determine the type of symptom produced, or by virtue of the failure of cross protection, that the Colorado red-node virus is not a strain of the yellow-bean mosaic virus. Field observations of red-node symptoms on mosaic-infected plants in Colorado in 1947 and 1948 supported the latter possibility.

---

1 Contribution from the Section of Botany and Plant Pathology, Colorado Agricultural Experiment Station, Fort Collins, Colorado. Published with the approval of the Director of the Colorado Agricultural Experiment Station as Scientific Journal Series Article No. 293 .

2 Associate Plant Pathologist.

Table 1. Symptoms produced by different bean varieties inoculated with the Colorado red-node virus.

Type of Symptom	Average number of plants expressing symptoms[a]																							
	Idaho Pinto No. 22	US No. 3 Refugee	US No. 5 Refugee	Michelite	Idaho Pinto	Great Northern UI No. 15	Bountiful	No. 181 California	Stringless Wax	Pinto No. 650	Kentucky Wonder White 765	Kentucky Wonder White 780	Kentucky Wonder Brown 814	Idaho Pinto No. 78	Idaho Pinto No. 111	Scottsbluff Pinto	Wyoming Pinto	San Juan Pinto Selection						
Reddening of veins	5	3	2	3	3	3	2	4	3	2	0	0	5	5	3	5	2							
Red lesions on stems and at nodes	2	1	0	3	3	1	1	0	0	0	0	0	2	2	2	3	1							

[a] Five replications of 5 plants each.

Literature Cited

1. Grogan, Roy G. A pod-distorting strain of the yellow-bean mosaic virus. Phytopath. 38:10. 1948 (Abstr.)

2. Skiles, R. L. and W. D. Thomas, Jr. Red node and shiny pod viroses of field beans. Proc. Colo.-Wyo. Acad. Sci. 3(6):44. 1948. (Abstr.)

3. Virgin, W. J. An unusual bean disease. Phytopath. 33:743-745. 1943.

4. Zaumeyer, W. M. and H. Rex Thomas. Greasy pod - a new virus of beans. Phytopath. 37:25. 1947. (Abstr.)

5. _____ Shiny pod (greasy pod) virus and its identity with black root virus. Phytopath. 38:29. 1948. (Abstr.)

COLORADO A & M COLLEGE, FORT COLLINS

CURLY TOP OF TOMATOES

R. D. Watson and J. E. Kraus

In The Country Gentleman last year an article was published on the basis of a report from Arizona that Burpee's Fordhook Hybrid tomato was resistant to curly top. In the Horticulture Department's variety comparison test at Lewiston, Idaho, it was found that Fordhook Hybrid was no more resistant than the other commercial varieties (see Table 1).

Table 1.
Horticulture Department Trial at Lewiston, Idaho
1946

Tomato Variety	Total No. Plants	No. Curly Top	Percentage Curly Top
1121 Fordhook Hybrid	43	5	11.6%
1132 Burpee Hybrid	45	6	13.3%
Sioux	45	5	11.1%
Early Stone	43	7	16.3%

Table 2.
Curly Top Test 1948

Tomato Variety	Caldwell		Jerome		Average Percentage Curly Top Caldwell & Jerome
	Total No.	Curly Top	Total No.	Curly Top	
1121 Fordhook Hybrid	10	7	16	16	89.0
1132 Burpee Hybrid	7	6	9	8	87.5
1133 Burpeeana Early Hybrid	9	7	16	16	92.0
1135 Clinton Hybrid	10	7	-	-	70.0
Commercial (check)	144	104	95	91	82. %

In view of these conflicting results a test was planned at our curly top breeding plots at Caldwell and Jerome, Idaho, using the Burpee hybrid tomatoes listed in the seed catalogue, Fordhook Hybrid being one of them. Results in Table 2 show that these hybrids are just as susceptible to curly top disease as are all other commercial tomatoes tested; they are susceptible to the strains of the curly top virus in Idaho.

The Burpee Company has never claimed any resistance to this disease in Fordhook Hybrid nor is the quality of the tomato considered here. For these reasons neither this company nor its product is involved in any way with this report. It seemed advisable to present the information as we find it in Idaho. If the Fordhook Hybrid continues to show resistance in Arizona, further investigation will determine the reason.

UNIVERSITY OF IDAHO, MOSCOW, IDAHO

THE OCCURRENCE OF RHIZOCTONIA CRATER ROT IN ILLINOIS-GROWN CARROTS

G. B. Ramsey and M. A. Smith

This disease has been known to occur in New York carrots since 1934.[1] Following serious losses in New York cold storages in 1942-1946, Rader[2] gave a complete description of the disease and named the causal organism Rhizoctonia carotae. Up to the present time, apparently, this organism has not been reported on carrots from any other State. This note is to report the occurrence of Rhizoctonia crater rot in epiphytotic form in carrots grown in Cook County, Illinois.

One of the large soup companies in Chicago stored about 22 million pounds of these carrots in various cold storages (32-34°F.) in and about Chicago, for processing during the winter and spring months (1948-49). The carrots removed from storage toward the latter part of February began to show small amounts of this decay, and during all of March the loss varied with different lots from 5 to 90 percent. The most severe loss occurred in top-layer baskets stored in a very humid room. In this room the baskets were wet and many of them were completely covered with the white, cottony mycelium of Rhizoctonia carotae. The carrots within these baskets showed up to practically 100 percent infection by this organism. The brown craters varied from 1/4 to 1 inch in diameter and from 1/8 to 1/2 inch deep. Most of these lesions showed white to cream-colored

---

1 Ramsey, G. B. Market pathology notes from Chicago. U. S. Dept. of Agr., Pl. Disease Reptr. 18: 40-41. 1934.

2 Rader, W. E. Rhizoctonia carotae n. sp. and Gliocladium aureum n. sp., two new root pathogens of carrots in cold storage. Phytopathology 38: 440-452. 1948.

Figure 1. Rhizoctonia crater rot of Illinois carrots.

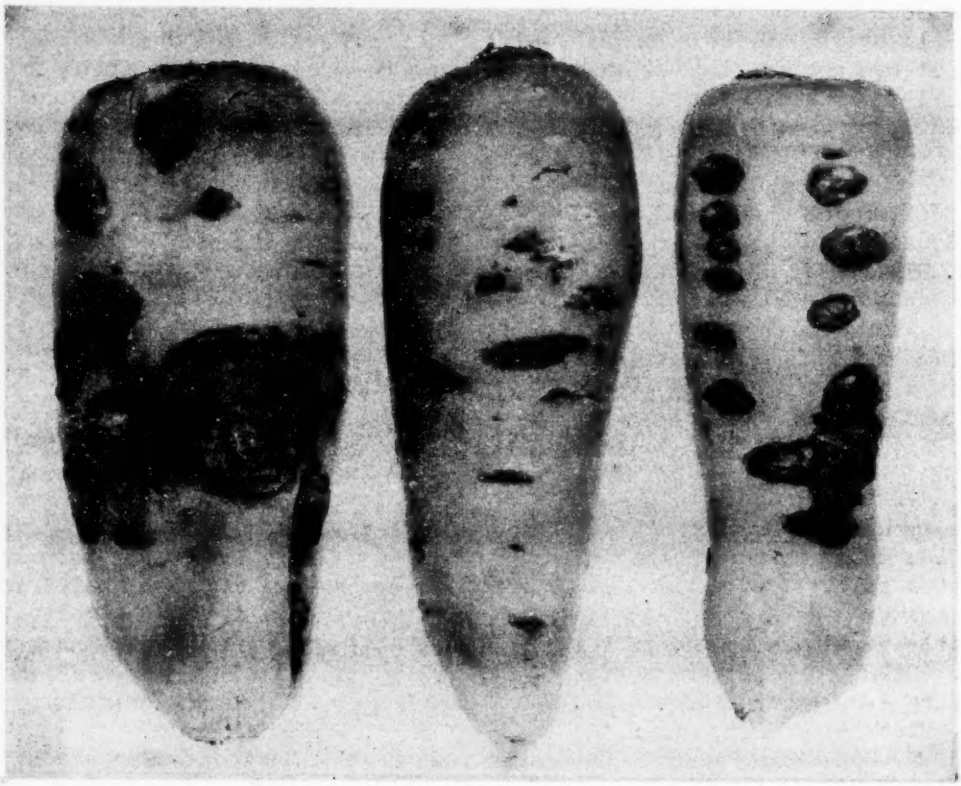


Figure 2. Crater rot on carrots that have been steamed for the removal of the epidermis. These lesions must be removed by hand paring before final processing.

mycelium on their surfaces, but no sclerotia were observed (Figs. 1 and 2). Other rots also present were caused by Botrytis, Alternaria, and Typhula; however, these were insignificant in comparison with the damage caused by the crater rot organism.

The soup company reports that their loss on account of this disease will run well over $100,000. Large numbers of baskets were so badly decayed that it was not feasible to process any of the carrots. In addition to the total loss of many baskets of carrots, the cost of processing the moderately affected roots was almost doubled. It was estimated that the extra labor cost for peeling and paring out the decayed spots amounted to about $5000 per week.

DIVISION OF FRUIT AND VEGETABLE CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AGRICULTURAL RESEARCH ADMINISTRATION, U. S. DEPARTMENT OF AGRICULTURE, CHICAGO, ILLINOIS

RESISTANCE OF WATERMELON VARIETIES TO FUSARIUM WILT

T. E. Smith and S. J. Hassell

The amount of Fusarium wilt on certain watermelon varieties was measured in 1948, using four replications of plots of ten hills located on naturally infested soil. Seed mainly from commercial sources were planted May 1 and wilt counts made August 13, after the melons had matured. The known susceptible varieties, Garrison, Dixie Queen, and Dude Creek, were 100 percent dead. Standard resistant varieties had wilt percentages as follows: Leesburg - 0.0, Hawkesbury - 10.3, Klondyke R7 - 12.5, Stone Mountain # 5 - 16.5, Blacklee - 18.5, and Kleckley's # 6 - 33.5. Experimental lines from the U. S. Vegetable Breeding Laboratory, Charleston, South Carolina, had wilt percentages as follows: W 290 - 0.0, W 297 - 11.3, W 322 - 11.8, W 308 - 2.5, and 47 # 3 - 78.3. Four different accessions of the Dixie Queen wilt-resistant melon were tested also. These averaged 6.0, 35.5, 40.0 and 100.0 percent wilt, showing considerable variation between seed sources. Kleckley # 6, with moderate resistance (33.5 percent wilt) produced a satisfactory crop of melons where the original stand was two to four plants per hill.

McNAIR'S YIELD-TESTED SEED COMPANY, LAURINBURG, NORTH CAROLINA

OCCURRENCE OF CABBAGE YELLOWS IN THE HASTINGS SECTION, FLORIDA

A. H. Eddins and Stanley M. Burrell

Cabbage yellows caused by Fusarium oxysporum f. conglutinans (Wr.) Snyder & Hansen was observed by the writers in cabbage fields in the Hastings area

for the first time in November 1948. Immediately after it was found, mimeographed copies of a description of the disease and information pertaining to its introduction, importance and means of control were prepared and mailed to plant pathologist and county agents in cabbage-growing areas of the State, and to cabbage growers in the Hastings area with a request that they assist in locating fields with plants showing symotoms of the disease. Fields in which it was reported to be present were inspected by the writers.

No reports of yellows in cabbage fields outside of the Hastings section were received during the 1948-1949 season. It was found at Hastings in 27 fields comprising approximately 313 acres. It destroyed 50 to 90 percent of the crop in 30 acres, 10 to 30 percent in 78 acres and 2 to 5 percent in 108 acres; a trace was seen in 97 acres. The disease may have been present in other fields but was not recognized or was not reported by the owners. The writers did not have time to inspect all fields comprising about 7,500 acres planted to cabbage in the Hastings area. The disease was scattered in some fields, with more plants showing infection in low-lying dark soil than in higher, lighter sand. It was uniformly distributed in other fields regardless of the lay of the land and the type of soil. The disease was found in yellows-susceptible Glory of Enkhuizen and Copenhagen Market varieties which were planted on 95 percent or more of the acreage. It was not seen in plantings of resistant Marion Market.

The first report of cabbage yellows in Florida was made by Weber, who found it in a winter crop of cabbage in the vicinity of Bartow, Florida, in October 1936.[1] He reported that it was present in several fields, and that it probably was introduced about 1920 in cabbage plants sent from Racine, Wisconsin for seed production. Apparently infested fields at Bartow have been planted to yellows-resistant varieties of cabbage or to non-susceptible crops since 1936, as the disease has not been reported as causing any damage there since that year.

No appreciable development of yellows occurs at soil temperatures below 60°F. or above 95°F. The winter of 1948-1949 was extremely mild in Florida and temperatures favored development of yellows in all cabbage-growing areas of the State.

The yellows organism was introduced into the Hastings section with affected seedling plants grown in yellows-sick soil in other States. During the last 10 years most of the early fall crops in the Hastings area have been set with plants imported mostly from Georgia. Plants grown in North Carolina, Tennessee, Virginia, and probably other States also have been used. The disease was most prevalent and destructive in fields set

---

[1] Weber, George F. Two new fungous diseases of vegetables in Florida. U. S. D. A. Plant Dis. Reptr. 15:282. 1937

with imported plants several years in succession. None was found in fields which have been set with plants grown locally in yellows-free soil. Just when the disease was first introduced and from what State or States it came is not known. However, reports from two growers indicate that it was introduced on one farm in Georgia-grown plants and on another with plants grown in the vicinity of Norfolk, Virginia. The writers saw the disease being carried into one field at Hastings in affected plants grown in yellows-sick soil on a nearby farm. There probably have been other cases of local distribution of the disease during the last two years.

FLORIDA AGRICULTURAL EXPERIMENT STATION, POTATO INVESTIGATIONS LABORATORY, HASTINGS

THE "CEPHALOTHECIUM DISEASE" OF CULTIVATED MUSHROOMS CAUSED BY A NEMATODE (DITYLENCHUS SP.), EVIDENCED BY SURFACE DEVELOPMENT OF PREDACEOUS FUNGI

E. B. Lambert, G. Steiner, and Charles Drechsler[1]

For several years a disease has been apparent in commercial mushroom beds, characterized by the disappearance of the mushroom mycelium in the beds and the development on the soil surface of a sparse fungus growth usually referred to Cephalothecium. As a rule, the disease is first noticed at about the time of the third flush in areas 3 to 6 feet in diameter. In typical cases the mushroom mycelium is completely destroyed in the affected areas and the compost acquires a fetid odor. The surface of the bed becomes sunken and irregular in the diseased areas as a result of the decomposition of the underlying compost and affected areas progressively encroach on the healthy portions of the beds.

The fact that a surface mold is always associated with the disease has led to the prevalent belief that the "Cephalothecium" apparent on the bed surface is causing the disintegration of the mushroom mycelium in the bed. On the other hand, the sparse nature of the fungus growth over the surface of the soil and the absence of a conspicuous fungus growth in the compost have raised doubts as to the extent of the damage caused by the fungus. Mites, springtails, and nemas from time to time have been suspected of consuming the mushroom mycelium; but here again the constant association of the fungus with the disease caused observers to hesitate to attribute the damage to these bed invaders. Consideration of the disease has remained in this stalemate for about ten years. Meanwhile the disease has been appearing in an increasing number of houses and has assumed considerable economic importance in the mushroom industry of eastern Pennsylvania. The identification by Drechsler of the surface molds as fungi normally predaceous on nemas suggested a re-evaluation and orientation of the role of the nemas and of the surface fungus in the "Cephalothecium" syndrome.

Compost was examined for the presence of nemas from diseased areas in nine separate mushroom establishments in Chester County, Pennsylvania, and in two near Hockessin, Delaware. In all cases an undescribed species of Ditylenchus has been observed feeding on the mushroom mycelium. The nema population in the compost varied considerably with the different collections; but whenever the samples were taken from portions of the bed that were evidently in the process of disease invasion the nemas were extremely numerous. The surface mold developing in diseased areas has

---

[1] Senior Mycologist, Division of Fruit and Vegetable Crops and Diseases, Principal Nematologist in Charge, Division of Nematology, and Pathologist, Division of Fruit and Vegetable Crops and Diseases, respectively. Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration.

been cultured and identified from three separate locations as Arthrobotrys superba Corda. Previous observers of this disease were undoubtedly collecting the same fungus but mistakenly referred it to Cephalothecium which resembles Arthrobotrys and has been frequently confused with it for nearly a century. It is anticipated that upon further study additional species of predaceous fungi will be found capturing the nemas.

All of the evidence at hand suggests that the symptoms usually associated with the Cephalothecium disease may be explained on the basis of the invasion of the beds by nematodes in sufficient numbers to consume the mushroom mycelium, followed by the development of predaceous fungi which prey upon the nemas and sporulate on the surface of the soil. The possible role of secondary microorganisms as aids in the disintegration of the mushroom mycelium must of course be taken into consideration. Observations of mushroom growers suggest that in some cases the nemas remain in the mushroom house between crops, presumably in the bed boards, while in other cases they are introduced with compost or casing soil.

The nematode is a new species related to the well-known Ditylenchus dipsaci. (A taxonomic description will be published elsewhere.) D. dipsaci is now considered a group of species most of which are plant pests attacking such hosts as hyacinth, narcissus, bulbous iris, tulip, phlox, hydrangea, alfalfa, clover, strawberry, rye, oats, potato, tobacco, sweetpotato, and many others. While all these species infect phanerogamic plants, the present one is the first that is apparently entirely mycophagus in its habits. It punctures the mushroom mycelium and feeds on the contents of the hyphae, destroying them directly or indirectly. Like other members of the genus the present species appears capable of rapid growth and propagation, eventually building up enormous numbers which migrate and spread through a mushroom bed and effect the destruction of the mycelium on an advancing front. If this new Ditylenchus is similar in behavior to its relatives, it should be able to go into dormancy during drought and other adverse environmental conditions and thus survive in dry debris and dry remains from infected mushroom beds for months or even several years. Such debris and remains are possibly the main source of reinfection. Investigations to determine this matter as well as to ascertain the life cycle of this new species and other facts of significance for an understanding of the present disease have been initiated. Methods of control are also being studied.

DIVISION OF FRUIT AND VEGETABLE CROPS AND DISEASES, AND DIVISION OF NEMATOLOGY, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

A ROOT ROT DISEASE OF ALFALFA CAUSED BY FUSARIUM SOLANI

Glen Staten and Philip J. Leyendecker

In 1945 at the New Mexico Experiment Station farms during a routine bacterial wilt[1] field evaluation study a substantial number of diseased alfalfa plants were encountered which did not exhibit normal bacterial wilt symptoms. External lesions as well as vascular discoloration were evident. In most cases the reddish brown vascular discoloration could be followed to the point of origin, which was in the majority of cases from lateral roots. Figure 2 shows the vascular discoloration, which is a dark red to cinnamon brown. Many infections remained localized in the crown area causing a typical crown rot. Figure 1 shows the fungus growing from the lesions of a diseased root that was placed in a moist chamber for 12 hours.

Numerous isolations were made and in each case a species of Fusarium was obtained. The fungus grew readily on Czapek's nitrate agar producing a fluffy white mycelium. Older cultures produced chlamydospores (blue) in abundance. Isolates were sent to Dr. W. C. Snyder at the University of California who determined the fungus to be Fusarium solani (Mart.) Appel & Wr.

Two methods were utilized to ascertain the pathogenicity of the organism. In one series, surface sterilized seed (variety, Buffalo) was planted in steamed soil infested with F. solani. Damping-off data and degree of vascular injury were recorded. Another series of pots containing artifically infested soil was planted with healthy transplants (roots 1/4 to 1/2 inch in diameter), which had been surface sterilized with 1/1000 $HgCl_2$. When the plants became well established the crowns were split and inoculated with the pathogen. In the seeded series of pots, 21 percent of the seedlings "damped off" and the fungus was readily reisolated from the diseased plants.

After 18 weeks, the remaining plants in both series (seeded and transplanted) were removed from the pots and the soils washed from the roots. Between 40 and 60 percent of the plants exhibited symptoms which had been previously noted in the field. Infected plants were stunted and the leaves appeared light green to yellow in color. Figure 3 shows the appearance of plants grown from seed 45 days after planting in infested (left) and non-infested (right) soil.

The pathogen seemed to be more virulent when entrance was afforded by natural or artificial wounding. Studies are now under way to determine the amount of inherent resistance which common alfalfa varieties and New Mexico Experiment Station selections may possess.

---

[1] Corynebacterium insidiosum (McCulloch) Jensen.

Figure 1

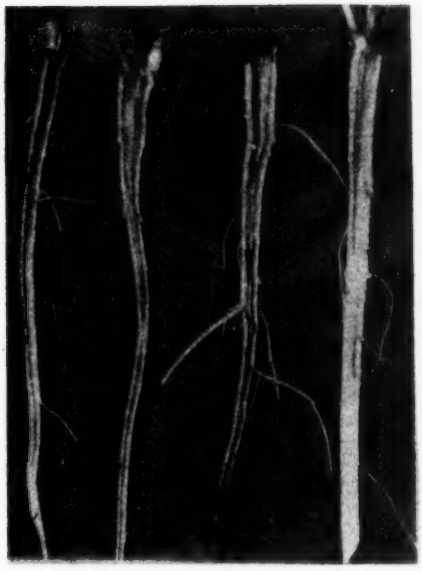


Figure 2

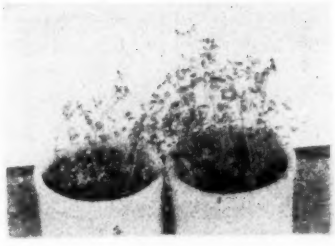


Figure 3

Fusarium solani root disease of alfalfa in New Mexico. (For explanation see text.)

References

Cormach, M. W. Fusarium spp. as root parasites of alfalfa and sweet clover in Alberta. Can. Jour. Res. Sec. C. 15: 493-510. 1937

Cottan, W. P. A "dry rot" disease of alfalfa roots caused by Fusarium. Phytopath. 11:383. 1921

Fergus, E. N. and Valleau, W. D. A study of clover failure in Kentucky. Kentucky Agr. Exp. Sta. Res. Bul. 269. 1926

Horsfall, J. G. A study of meadow crop diseases in New York. New York Agr. Exp. Sta. Mem. 130. 1930

Weimer, J. L. A wilt disease of alfalfa caused by Fusarium sp. Phytopath. 17:337-338. 1927

_____ A wilt disease of alfalfa caused by Fusarium oxysporum var. medicaginis, n. var. Jour. Agr. Res. 37:419-433. 1928

_____ Additional hosts of Fusarium oxysporum var. medicaginis. Jour. Agr. Res. 39:351-353. 1929

DEPARTMENTS OF AGRONOMY AND BIOLOGY, NEW MEXICO AGRICULTURAL EXPERIMENT STATION, STATE COLLEGE

ALFALFA-CLOVER DISEASE SURVEY IN VIRGINIA

S. B. Fenne, R. G. Henderson, T. J. Smith, G. M. Shear

During the period of March 21 to 25 alfalfa and clover fields were visited in James City, Henrico, Orange, Culpeper, Fauquier, and Fairfax Counties. The writers were accompanied by C. L. Lefebvre and J. L. Allison of the Bureau of Plant Industry, Soils, and Agricultural Engineering.

Alfalfa

Stem rot, Sclerotinia trifoliorum, was readily found on alfalfa in most of the areas visited except in Orange County. For several years Sclerotinia has been the cause of serious losses to alfalfa in Eastern Virginia. In one planting on the experiment station plots at Williamsburg the new Williamsburg variety of alfalfa showed approximately 10% kill from stem rot. This variety, which was developed in eastern Virginia, survives for a longer period of time than all other varieties tested and previous observations suggested that it was partially resistant to stem rot. In the experimental plantings at the Station it was very striking to observe the apparently poor adaptation of certain varieties. Grimm, Ladak, and

Turkestan varieties were very poor in contrast to Atlantic, Kansas Common and Williamsburg. Stands went out during the summer and fall months, due apparently to a combination of anthracnose, Colletotrichum trifolii, black stem, Ascochyta imperfecta, and Stemphylium leaf spot S. botryosum.

Black Stem was found generally distributed in almost every alfalfa field examined and in many instances had already caused severe injury. Black stem was especially destructive on new stands of alfalfa. In 1948, in many parts of the State, black stem was very destructive and in fields the first cutting of alfalfa was practically worthless.

Stem nematode, Ditylenchus sp., was found widespread on one farm in Henrico County. The disease had been observed on this farm in 1948. The disease was more severe this year and apparently was responsible for killing out the stands of susceptible varieties. Smith and Henderson seeded plots on this farm last August to a number of nematode resistant strains. Stands of several strains, including the Nemastan variety, were practically destroyed by the stem nematode disease and the prevalent fungus diseases. The most promising selection appeared to be 77-46 and that had only a 75% stand. In addition, the following strains appeared almost as good: 77-98, 77-97, 77-113, 11-C84. All of the standard varieties in the test were very poor. Fusarium sp. was isolated from plants collected in this field in 1948 and it is probable that this organism was responsible for some of the damage on the old stand. Sclerotinia was also observed causing considerable damage.

Rhizoctonia solani was isolated from several collections made on this trip. In some cases seedlings were being killed. Brown, dead shoots were evident which were probably killed last fall but were still covered with hyphae of Rhizoctonia. Pseudopeziza trifolii, Stemphylium botryosum and Peronospora trifoliorum, were found in several fields visited.

Bacterial wilt, Corynebacterium insidiosum, was found in James City and Henrico Counties. In both of these cases the alfalfa had died out probably due in most part to bacterial wilt.

Some interesting "date-of-planting" plots were observed at the Experiment Station at Williamsburg, in which alfalfa was sown on different dates from June to October in 1948. The July seeding was best in appearance, followed closely by that made in August. The September seeding was fair while the October seeding was very poor with only a few small plants remaining. The plantings made in May and June had good stands but were very weedy. Sclerotinia appears to cause much more damage on late plantings of alfalfa and, therefore, in order to escape serious injury from stem rot it is recommended that alfalfa be seeded between July 15 and August 15, also that alfalfa be planted on soil that has not grown clover or alfalfa during the preceding two or three years.

Clover

Stem rot, Sclerotinia trifoliorum, was readily found on Crimson and Red clovers, and in Culpeper County it was causing serious injury on Ladino clover. The disease occurred in spots a foot or more in diameter on which all the plants were killed. In general Ladino was less seriously affected by stem rot than other clovers.

It is difficult to explain why in some areas alfalfa was injured by Sclerotinia and Ladino clover was free from injury, while in other areas the reverse was true. Parasitic races may help explain this situation. On several fields visited in Fauquier county, Ladino clover was observed dying out. Decaying stolons that appeared soft and watery suggested injury by Sclerotinia, but no sclerotia were observed. All of these fields had been grazed heavily and, compared to others we saw, they had not been adequately fertilized.

The most severe injury caused by Sclerotinia on the entire trip was observed on a legume-grass farm in Fairfax County. Most of this 300-acre farm is kept in Ladino clover and orchard grass mixture. It has been the custom to grow this mixture on a field for five or six years and then disc it up and reseed. In some fields alfalfa is included in the mixture. It appears that by following this legume-grass mixture program, Sclerotinia inoculum has been encouraged and built up to the point that in some fields patches of Ladino clover six feet or more in diameter have been killed. Sclerotia lying on the surface of the ground could be literally picked up by the handful.

In the clover plots at the Williamsburg Experiment Station certain varieties, especially those of Western origin showed poor growth and thin stands. Several strains of the Virginia Northern Neck variety were outstanding, while Kenland, Cumberland, Midland, and Tennessee Purple varieties were in an intermediate class between the Northern Neck strains and the Western varieties.

EXTENSION SERVICE, VIRGINIA POLYTECHNIC INSTITUTE, BLACKSBURG

RICE DISEASES IN ARKANSAS IN 1948

E. M. Cralley and C. Roy Adair

Stem rot (Leptosphaeria salvinii Catt.). Stem rot caused moderate to heavy damage in certain areas in 1948. The disease was especially serious on certain long-grained varieties such as Fortuna, Nira, and Bluebonnet. Heavy applications of nitrogenous fertilizers tend to increase the severity of the disease. Rice farmers in Lonoke County reported satisfactory control of stem rot when potash fertilizer was used in combination with

nitrogenous fertilizer (2).

Seedling diseases (Helminthosporium oryzae Breda de Haan, Fusarium spp., Rhizoctonia solani Kuehn, and Pythium spp.). Rice seedling blight is a disease complex encountered in all of the rice-growing areas of the State. Most farmers reported satisfactory results from seed treatment of early sown rice. Experiments at the Rice Branch Station at Stuttgart showed that Ceresan M gave somewhat better stands than did Arasan, Yellow Cuprocide, or Phygon when the seed was drilled, but Yellow Cuprocide was definitely superior to Arasan or Phygon when the seed was sown in water. Treatments with combinations of the above-mentioned seed disinfectants have shown no advantage over individual treatments.

Straighthead (Sterility). Straighthead caused serious losses in a number of areas in 1948. The most severe losses were observed on pasture land that was cropped to rice for the first time in 1948. In one 200 acre field the yield was reduced by about 50 bushels per acre, causing a total loss of at least 20,000 dollars. Thorough drainage of the soil has, in general, given satisfactory control of straighthead. The time of draining is an important control factor and severe losses from straighthead have been observed when draining was done too late in the growing season.

There appears to be a difference in the susceptibility of rice varieties to straighthead. In an outlying varietal test at Clarendon, straighthead was light on Caloro, Asahi, and Prelude; moderately severe on Zenith, Arkansas Fortuna, Nira 43, and Bluebonnet; and very severe on Magnolia, Kamrose, Arkrose, Blue Rose 41, and Rexark.

Leaf spot (Cercospora oryzae Miyake). A survey of the rice-growing areas showed that leaf spot was light to moderate in the northern and central rice growing areas but was very severe in the southern areas. Certain strains of the fungus are obviously increasing in importance in the State (1,4). Blue Rose 41, Magnolia, Prelude, Arkansas Fortuna, Bluebonnet, Rexora X Nira hybrid (B40-9), and Nira, which heretofore had been considered resistant to moderately resistant, were severely infected in certain localities. Asahi, Kamrose, Arkrose selection 44-1, and Hill Long Grain were the only varieties that were uniformly resistant in the five areas tested.

White tip. White tip was observed throughout the rice-growing areas. The symptoms are very similar to those of a disease caused by a nematode. Aphelenchoides oryzae Yokoo, observed in Japan in 1947 (3). Experiments at the Rice Branch Station show that Arkansas Fortuna, Nira 43, Bluebonnet, and Hill Long Grain are resistant to white tip. Experiments on the effect of date of planting have shown that the severity of white tip is decreased when the rice is sown early in the season.

Black sheath rot (Ophiobolus oryzinus Sacc.). Black sheath rot has been observed in many of the rice-growing areas of the State and is ordinarily considered to be of minor economic importance. However, during 1946-48, the disease caused serious losses in the southern part (Drew County). Black sheath rot may cause severe lodging and farmers often mistake it for stem rot. Observations in 1947 (5) indicate that Cody, Asahi, Zenith, Kamrose, Arkansas Fortuna, Nira 43, Bluebonnet, and Hill Long Grain may be resistant to moderately resistant to this disease.

Literature Cited

1. Chilton, S. J. P., and Tullis, E. C. A new race of Cercospora oryzae on rice. Phytopath. 36:950-952. 1946

2. Cralley, E. M. Rich nutrition in relation to stem rot of rice. Better Crops with Plant Food. 31 (4). 1947

3. _____ White tip of rice. Phytopath. (Abs.) Phytopath. 39:5. 1949

4. Ryker, T. C. Physiologic specialization in Cercospora oryzae. Phytopath. 33:70-74. 1943

5. Tullis, Edgar C. and Adair, C. Roy. Black sheath rot of rice (Ophiobolus oryzinus) caused lodging of rice in Arkansas and Texas in 1947. Plant Dis. Rptr. 31 (14):468. 1947

COOPERATIVE INVESTIGATIONS BY ARKANSAS AGRICULTURAL EXPERIMENT STATION AND THE DIVISION OF CEREAL CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AGRICULTURAL RESEARCH ADMINISTRATION, U. S. DEPARTMENT OF AGRICULTURE

SOME NEW DISEASE RECORDS OF GRAMINEAE IN THE WESTERN UNITED STATES-III[1]

Roderick Sprague and Jack P. Meiners[2]

Some of these additions to the state-by-state host range of leafspots and rusts on Gramineae were determined, from 1948 collections in the

[1] Cooperative investigations between the Washington Agricultural Experiment Station and the Division of Forage Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Department of Agriculture. Published with the approval of the Director of the Washington Agricultural Experiment Station. Research Note No. 5.

[2] Pathologist, Division of Plant Pathology, Washington Agr. Experiment Station; and formerly, Agent, Division of Forage Crops and Diseases (now Assoc. Pathologist, Div. of Hort. Crops and Diseases, U. S. Department of Agriculture) U. S. Dept. of Agr., respectively.

interim since the last report was submitted.[3] Others given here are accumulations from earlier determinations which have not been reported previously in this series.

AEGILOPS CYLINDRICA Host.

Fusarium acuminatum Ell. & Ev., Pullman, Wash.
Puccinia rubigo-vera (DC.) Wint., Pullman, Wash.
Pythium debaryanum Hesse, Pullman, Wash.

AGROPYRON CANINUM (L.) Beauv.

Fusarium acuminatum Ell. & Ev., Pullman, Wash.

AGROPYRON CRISTATUM (L.) Gaertn.

Fusarium acuminatum Ell. & Ev., Moorcroft, Wyo.
Ovularia pusilla (Ung.) Sacc. and D. Sacc., Ten miles s. of Craig, Colo.
Pseudomonas coronafaciens var. atropurpureum (Reddy & Godkin) Stapp, Moorcroft, Wyo.
Scolecotrichum graminis Fckl., Ft. Totten, N. Dak.
Selenophoma donacis (Pass.) Sprague & A. G. Johnson, Libby Creek in Medicine Bow Natl. For., Wyo.
Septoria avenae f. sp. triticea T. Johnson, Bismarck, N. Dak.

AGROPYRON GRIFFITHSII Scribn. & Sm.

Fusarium acuminatum Ell. & Ev., Ekalaka, Mont.
Pseudomonas coronafaciens var. atropurpureum (Reddy & Godkin) Stapp, Ekalaka, Mont.; Belfield, N. Dak.
Scolecotrichum graminis Fckl., Harney Natl. For., S. Dak.

AGROPYRON INERME (Scribn. & Sm.) Rydb.

Claviceps purpurea (Fr.) Tul., Mandan, N. Dak.
Erysiphe graminis DC., Baker Hole, Mont.
Puccinia montanensis Ellis, Logan Canyon, Utah
P. rubigo-vera (DC.) Wint., Carey, Ida.
Scolecotrichum graminis Fckl., Carey, Ida.; Wind River Canyon, Wyo.

AGROPYRON INTERMEDIUM (Host.) Beauv.

Helminthosporium tritici-repentis Died., Mandan, N. Dak.

AGROPYRON REPENS (L.) Beauv.

Claviceps purpurea (Fr.) Tul., Pullman, Wash.
Helminthosporium tritici-repentis Died., four miles s. of Wilkeson, Wash.

---

[3] Sprague, R. and Meiners, Jack P. Some new disease records of Gramineae in the western United States. U. S. Bureau of Plant Industry, Soils, and Agricultural Engineering Plant Dis. Reptr. 33(3):147-158. Mar. 15, 1949.

Selenophoma donacis (Pass.) Sprague and A. G. Johnson, Pingree, N. Dak.
Septoria agropyrina Lobik, Cotton Lake, Minn.; Ft. Totten, N. Dak.; Brookings, Vermillion, S. Dak.
Septoria nodorum Berk., Carrington, N. Dak.

AGROPYRON RIPARIUM Scribn. & Sm.

Claviceps purpurea (Fr.) Tul., Mandan, N. Dak.
Fusarium acuminatum Ell. & Ev., Tensleep, Wyo.
Pseudomonas coronafaciens var. atropurpureum (Reddy & Godkin) Stapp, eastern Mont., adjacent N. Dak. and eastern Wyo.
Scolecotrichum graminis Fckl., New England, N. Dak.; Sundance, Wyo.
Selenophoma donacis (Pass.) Sprague & A. G. Johnson, Mandan, N. Dak.; Four Corners, Wyo.

AGROPYRON SMITHII Rydb.

Ascochyta agropyrina (Fairm.) Trotter, Yates, Forsythe, Mont.; Glen Ullen, N. Dak.
Fusarium moniliforme Sheld., Valentine, Nebr.
Helminthosporium tritici-repentis Died., Alzada, Mont.; Vermillion, S. Dak.; Colony, Wyo.
Pseudomonas coronafaciens var. atropurpureum (Reddy & Godkin) Stapp, eastern Mont.; Bassett, Valentine, Nebr.; Wyo.
Scolecotrichum graminis Fckl., Valentine, Nebr.; Buffalo, S. Dak.; Four Corners, Wyo.
Septoria elymi Ell & Ev., Valentine, Nebr.
S. nodorum Berk., Belfield, N. Dak.

AGROPYRON SPICATUM (Pursh) Scribn. & Sm.

Erysiphe graminis DC., ten miles s. of Mabton, Wash.
Pseudomonas coronafaciens var. atropurpureum (Reddy & Godkin) Stapp, Tensleep Canyon, Wyo.
Puccinia rubigo-vera (DC.) Wint., near Deary, Ida.
Septoria elymi Ell. & Ev., Sundance, Wyo.

AGROPYRON SUBSECUNDUM (Lk.) Hitchc.

Scolecotrichum graminis Fckl., Wilton, N. Dak.
Septoria elymi Ell. & Ev., Waterloo, Mont.; Wilton, N. Dak.

AGROPYRON TRACHYCAULUM (Lk.) Malte

Ascochyta agropyrina (Fairm.) Trotter, Upper Sheep Creek (Uinta Mts.), Utah
Bacterium agropyri (O'Gara) Stapp, Hamilton, Colo.
Selenophoma donacis (Pass.) Sprague & A. G. Johnson, Libby Creek in Medicine Bow Natl. For., Wyo.
Selenophoma obtusa Sprague & A. G. Johnson, Libby Creek, Medicine Bow Natl. For., Wyo.

AGROSTIS ALBA L.

Colletotrichum graminicola (Ces.) Wils., S. F. of Clearwater River, Ida.
Helminthosporium stenacrum Drechsl., Bearmouth, Mont.; Donnelly, Ida.
Rhizoctonia solani Kuehn, Merriman, Nebr.
Spermospora subulata f. nov. Sprague, Teton Pass, Wyo.

AGROSTIS EXARATA Trin.

Colletotrichum graminicola (Ces.) Wils., five miles s. of Rochester, Wash.
Scolecotrichum graminis Fckl., Camel's Hump Pass (Hwy. 10), Mont.

AGROSTIS EXARATA var. AMPLA (Hitchc.) Hitchc.

Puccinia coronata Corda, eight miles e. of Sweet Home, Oreg.
Scolecotrichum graminis Fckl., Sweet Home, Oreg.

AGROSTIS OREGONENSIS Vasey

Scolecotrichum graminis Fckl., Camel's Hump Pass, Mont.

AGROSTIS PALUSTRIS Huds.

Corticium fuciforme (Berk.) Wakef., Pullman, Wash.
Typhula itoana Imai, Pullman, Wash.

AGROSTIS ROSSAE Vasey

Scolecotrichum graminis Fckl., Chinook Pass, Wash.

AGROSTIS SCABRA Willd.

Cercospora agrostidis Atk., Mullen Spring, Ida.
Helminthosporium stenacrum Drechsl., Mullen Spring, Ida.
Selenophoma everhartii (Sacc. & Syd.) Sprague & A. G. Johnson, Fox Ridge, S. Dak.

AGROSTIS SCABRA var. GEMINATA (Trin.) Swallen

Scolecotrichum graminis Fckl., Medicine Bow Natl. For., Wyo.

ANDROPOGON FURCATUS Muhl.

Helminthosporium sp. (buchloes), Newcastle, Wyo.
Septoria andropogonis Davis, Merriman, Nebr.; Wewela, Martin, S. Dak.

ANDROPOGON SCOPARIUS Michx.

Septoria andropogonis J. J. Davis, Eli, Nebr.

ANTHOXANTHUM ODORATUM L.

Helminthosporium dematioideum Bub. & Wrob., five miles s. of Rochester, Wash.

ARISTIDA LONGISETA Steud.

Fusarium acuminatum Ell. & Ev., Long Pine, Nebr.; Shoshoni, Wyo.
Helminthosporium sp. (buchloes), Wind River Canyon, Wyo.

ARISTIDA OLIGANTHA Michx.

Selenophoma everhartii (Sacc. & Syd.) Sprague & A. G. Johnson, Albia, Iowa.

BOUTELOUA CURTIPENDULA (Michx.) Torr.

Helminthosporium sp. (buchloes), relatively common in N. Dak., S. Dak., adjacent Nebr., Newcastle, Wyo.
Septoria andropogonis J. J. Davis, Wewela, S. Dak.

BOUTELOUA GRACILIS (H.B.K.) Lag.

Helminthosporium sp. (buchloes), Belltower, Mont.; Valentine, Nebr.; Buffalo, Camp Crook, Neibar, S. Dak.; Colony, Wyo.

BROMUS CARINATUS Hook. & Arn.

Scolecotrichum graminis Fckl., Cheyenne Crossing, S. Dak.

BROMUS CILIATUS L.

Scolecotrichum graminis Fckl., Silver Plume, Colo.

BROMUS JAPONICUS Thunb.

Helminthosporium bromi Died., Alzada, Mont. and adjacent Wyo.; s. of Ft. Pierre, S. Dak.
Septoria bromi Sacc., Carns, Nebr.; Moskee, Wyo.

BROMUS LAEVIPES Shear

Scolecotrichum graminis Fckl., west of St. Regis, Mont.

BROMUS PUMPELLIANUS Scribn.

Fusarium avenaceum (Fr.) Sacc., Big Horn Mts., Wyo.
Pseudomonas coronafaciens var. atropurpureum (Reddy & Godkin) Stapp, Black Hills, S. Dak.; Black Hills, Big Horn Mts., Wyo.
Rhynchosporium secalis (Oud.) J. J. Davis, Big Horn Mts., Wyo.
Rhizoctonia solani Kuehn, Big Horn Mts., Wyo.

BROMUS SECALINUS L.

Claviceps purpurea (Fr.) Tul., Pullman, Wash.

BROMUS SITCHENSIS Trin.

Scolecotrichum graminis Fckl., Whidby Is., Wash.

BROMUS TECTORUM L.

Ascochyta sorghi Sacc., Detroit Lake, Minn.

BROMUS VULGARIS (Hook.) Shear

Stagonospora bromi Sm. and Ramsb., S. F. Clearwater River, Ida.

CALAMAGROSTIS CANADENSIS (Michx.) Beauv.

Mastigosporium rubricosum (Dearn. and Barth. apud. Dearn.) Nannf., Sheep Creek (Uinta Mts.) Utah; Pipestone Pass, Mont. (reported from Mont. on var. scabra, type of Fusoma rubricosum, but not on species proper.)
Scolecotrichum graminis Fckl., Skyway, Colo.; Republic, Wash.
Septoria avenae Frank, Baker Hole, Mont.

CALAMAGROSTIS INEXPANSA A. Gray

Colletotrichum graminicola (Ces.) Wils., Twin Bridges, Mont.

CALAMAGROSTIS MONTANENSIS Scribn.

Pseudomonas coronafaciens (Elliott) Stapp, Camp Crook, S. Dak.
Rhizoctonia solani Kuehn, Camp Crook, S. Dak.

CALAMAGROSTIS PURPURASCENS R. Br.

Colletotrichum graminicola (Ces.) Wils., Tarkio, Mont.

CALAMOVILFA GIGANTEA (Nutt.) Scribn. & Merr.

Puccinia amphigena Ktze., Belltower, Mont.

CALAMOVILFA LONGIFOLIA (Hook.) Scribn.

Septoria calamovilfae Petr. Glendive, Mont.; Colony, Wyo.

CATABROSA AQUATICA (L.) Beauv.

Erysiphe graminis DC., Sand Coulee, Mont.; Rough Lock Falls, S. Dak.
Puccinia poae-sudeticae (West.) Jørstd., Rough Lock Falls, S. Dak.

DACTYLIS GLOMERATA L.

Puccinia rubigo-vera (DC.) Wint., Bozeman, Mont.
Rhynchosporium secalis (Cud.) J. J. Davis, Camel's Hump Pass, Mont.

DANTHONIA INTERMEDIA Vasey

Stagonospora subseriata (Desm.) Sacc., above Skyway, Colo., near summit in sub-alpine to alpine area; associated with Leptosphaeria sp.

DESCHAMPSIA DANTHONIOIDES (Trin.) Munro

Selenophoma donacis var. stomaticola (Bauml.) Sprague & A. G. Johnson, Grangeville, Ida.

DESCHAMPSIA ELONGATA (Hook.) Munro

Helminthosporium sp., Camel's Hump Pass, Mont.
Scolecotrichum graminis Fckl., Camel's Hump Pass, Mont.
Septoria nodorum Berk., Mt. Rainier, Wash.

ELYMUS ANTARCTICUS Hook. f.

Puccinia graminis Pers., Mandan, N. Dak.
P. montanensis Ell., Mandan, N. Dak.

ELYMUS ARISTATUS Merr. (Hybrid)

Septoria agropyrina Lobik, S. Fork of Clearwater River, Ida.

ELYMUS CANADENSIS L.

Pseudomonas coronafaciens var. atropurpureum (Reddy & Godkin) Stapp, Belltower, Wibaux, Yates, Mont.; Big Horn Mts., Wyo.
Rhizoctonia solani Kuehn, Eli, Nebr.
Septoria agropyrina Lobik, Belle Fourche, S. Dak.

ELYMUS CONDENSATUS Presl.

Scolecotrichum graminis Fckl., five miles s. of Craig, Colo.
Septoria infuscans (Ell. & Ev.) Sprague, five miles s. of Craig, Colo.
Puccinia rubigo-vera (DC.) Wint., Prosser, Wash.

ELYMUS DAHURICUS Turcz.

Claviceps purpurea (Fr.) Tul., Mandan, N. Dak.

ELYMUS EXCELSUS Turcz.

Xanthomonas translucens L. R. Jones, A. G. Johnson, & Reddy (Hagb.) Wallin, Mandan, N. Dak.

ELYMUS GIGANTEUS Vahl

Septoria agropyrina Lobik, Mandan, N. Dak.

ELYMUS GLAUCUS Buckl.

Ascochyta sorghi Sacc., S. F. Clearwater R., Ida.
Septogloeum oxysporum Sacc., Bomm. and Rouss., S. F. Clearwater R., Ida.; Teton Pass, Wyo.
Septoria agropyrina Lobik, Rochester, Wash.

ELYMUS INNOVATUS Beal

Rhynchosporium secalis (Oud.) J. J. Davis, Black Hills, S. Dak.

ELYMUS MACOUNII Vasey (hybrid)

Scolecotrichum graminis Fckl., s. of Bismarck, N. Dak.; Worland, Wyo.

ELYMUS SIBIRICUS L.

Pseudomonas coronafaciens var. atropurpureum (Reddy & Godkin) Stapp, Mandan, N. Dak.
Septoria nodorum Berk., Mandan, N. Dak.
S. avenae f. sp. triticea T. Johnson, Mandan, N. Dak.

FESTUCA ELATIOR L.

Helminthosporium dictyoides Drechsl., Gooding, Ida.

FESTUCA IDAHOENSIS Elmer

Claviceps purpurea (Fr.) Tul., Mack's Inn, Ida.; ten miles n. of Willowdale, Oreg.
Hendersonia graminis McAlp., north of Redmond, Oreg.

FESTUCA OCCIDENTALIS Hook.

Phoma sp. (black stem) St. Regis, Mont.

FESTUCA RUBRA L.

Colletotrichum graminicola (Ces.) Wils., Rochester, Wash.
Scolecotrichum graminis Fckl., Rochester, Wash.
Spermospora subulata (Sprague) Sprague, Rochester, Wash.

GLYCERIA GRANDIS S. Wats.

Scolecotrichum graminis Fckl., Doyon, N. Dak.

HIEROCHLOA ODORATA (L.) Beauv.

Rhizoctonia solani Kuehn, Big Horn Mts., Wyo.

HORDEUM BREVISUBULATUM (Trin.) Lk.

Selenophoma donacis var. stomaticola (Bauml.) Sprague & A. G. Johnson, Mandan, N. Dak.
Xanthomonas translucens var. cerealis (Hagb.) Wallin, Mandan, N. Dak.

HORDEUM MURINUM L.

Septoria avenae f. sp. triticea T. Johnson, twelve miles n. of Madras, Oreg.

HORDEUM NODOSUM L.

Claviceps purpurea (Fr.) Tul., Pullman, Wash.

HORDEUM PUSILLUM Nutt.

Rhizoctonia solani Kuehn, Albion, Mont.; Riverview, Nebr.; Usta, S. Dak.
Scolecotrichum graminis Fckl., Faith, Usta, S. Dak.; Albion, Glendive, Mont.

KOELERIA CRISTATA (L.) Pers.

Curvularia geniculata (Tracy & Earle) Boed., Yates, Mont.
Helminthosporium avenae Eidam, Buffalo, S. Dak.
Pseudomonas coronafaciens (Elliott) Stapp, New York Mills, Minn.
Rhizoctonia solani Kuehn, Benson, Minn.
Scolecotrichum graminis Fckl., Wibaux, Mont.
Septoria calamagrostidis f. koeleriae (Cocc. & Mor.) Sprague, Smith Ferry, Ida.

MELICA BULBOSA Geyer

Septoria nodorum Berk., Logan Canyon, Utah

MUHLENBERGIA FILIFORMIS (Thurb.) Rydb.

Scolecotrichum graminis Fckl., McCall, Ida.

MUHLENBERGIA PUNGENS Thurb.

Fusarium acuminatum Ell. & Ev., Eli, Nebr.

MUHLENBERGIA RACEMOSA (Michx.) B.S.P.

Cercospora muhlenbergiae Atk., Wilton, N. Dak.
Fusarium poae (Pk.) Wr., Washkish, Minn.
Rhizoctonia solani Kuehn, Lake Itasca, Minn.; Milbank, S. Dak.

MUHLENBERGIA SQUARROSA (Trin.) Rydb.

Phyllachora vulgata Th. & Syd. Twin Bridges, Mont.

CRYZOPSIS HYMENOIDES (Roem. & Schult.) Rick.

Bends, cause unknown. Glendive, Mont.
Scolecotrichum graminis Fckl., Four Corners, Wyo.

PHALARIS ARUNDINACEA L.

Rhynchosporium secalis (Oud.) J. J. Davis, West Yellowstone, Mont.

PHLEUM ALPINUM L.

Scolecotrichum graminis Fckl., Togwatee Pass, Wyo.

PHLEUM PRATENSE L.

Heterosporium phlei Gregory, Sundance, Wyo.

POA ALPINA L.

Erysiphe graminis DC., Medicine Bow Natl. For., Wyo.
Selenophoma donacis (Pass.) Sprague & A. G. Johnson, Skyway, Colo.

POA AMPLA Merr.

Claviceps purpurea (Fr.) Tul., Mandan, N. Dak.
Erysiphe graminis DC., Glenrock, Letter, Wyo.
Ovularia pusilla (Ung.) Sacc. & D. Sacc., Victor, Ida.; Teton Pass, Wyo.
Puccinia poae-sudeticae (West.) Jørstd., Uinta Mts., Utah
Rhizoctonia solani Kuehn, Yates, Mont.; Beach, N. Dak.; Glenrock, Wyo.
Selenophoma donacis (Pass.) Sprague & A. G. Johnson, Capitol, Mont.; Glenrock, Wyo.
Septoria oudemansii Sacc., Mandan, N. Dak.; Custer Natl. For., Mont.

POA ARACHNIFERA Torr.

Puccinia graminis Pers., Mandan, N. Dak.
Septoria oudemansii Sacc., Mandan, N. Dak.

POA ARIDA Vasey

Claviceps purpurea (Fr.) Tul., Mandan, N. Dak.
Helminthosporium vagans Drechsl., Mandan, N. Dak.
Scolecotrichum graminis Fckl., eastern Mont.

POA ARCTICA R. Br.

Fusarium nivale (Fr.) Ces., Medicine Bow Natl. For., Wyo.

POA CANBYI (Scribn.) Piper

Colletotrichum graminicola (Ces.) Wils., Mason, S. Dak.
Erysiphe graminis DC., Sheep Creek, Utah; Loyd, Colo.
Scolecotrichum graminis Fckl., Mason, S. Dak.
Uromyces dactylidis Otth, Umatilla Natl. For., Wash.

POA COMPRESSA L.

Claviceps purpurea (Fr.) Tul., Long Beach, Wash.
Colletotrichum graminicola (Ces.) Wils., Detroit Lake, Minn.
Rhizoctonia solani Kuehn, Frazee, Minn.; Mandan, N. Dak.

POA NERVOSA (Hook.) Vasey

Erysiphe graminis DC., Uinta Mts., Wyo.

POA PALUSTRIS L.

Selenophoma donacis (Pass.) Sprague & A. G. Johnson, Skyway Point, Colo.
Septoria oudemansii Sacc., Skyway, Colo.

POA SECUNDA Presl

Claviceps purpurea (Fr.) Tul., S. F. Clearwater R., Ida.
Septoria oudemansii Sacc., S. F. Clearwater R., Ida.

SECALE CEREALE L.

Fusarium nivale (Fr.) Ces., Mandan, N. Dak.; Wenatchee, Wash.
Hendersonia graminis McAlp., Ekalaka, Mont.

SETARIA VERTICILLATA (L.) Beauv.

Fusarium acuminatum Ell. & Ev., Denver, Colo.
Phyllosticta sorghina Sacc., Denver, Colo.

SETARIA VIRIDIS (L.) Beauv.

Pseudomonas syringae Van Hall, Crane, Mont.
Rhizoctonia solani Kuehn, Springview, Nebr.; Eakin, S. Dak.

SITANION HYSTRIX (Nutt.) J. G. Sm.

Puccinia graminis Pers., Tensleep, Wyo.
P. pattersoniana Arth., Logan Canyon, Utah.
Rhizoctonia solani Kuehn, Newcastle, Shoshoni, Sundance, Wyo.
Scolecotrichum graminis Fckl., Shoshoni, Tensleep, Wyo.

STIPA COLUMBIANA var. NELSONII (Scribn.) Hitchc.

Claviceps purpurea (Fr.) Tul., Colfax, Wash.
Scolecotrichum graminis Fckl., Baker Hole, Mont.; Colfax, Wash.
Septoria secalis var. stipae Sprague, seven miles up creek from Colfax, Wash.
Stagonospora simplicior var. stipicola Sprague, Uinta Mts., Utah.

STIPA COMATA Trin. & Rupr.

Pseudomonas coronafaciens var. atropurpureum (Reddy & Godkin) Stapp, Belltower, Baker, Ekalaka, Glendive, Mont.; Bassett, Nebr.; Buffalo, Camp Crook, S. Dak.; Beulah, Wyo.
Scolecotrichum graminis Fckl., Bradley, S. Dak.; Sundance, Wyo.
Selenophoma donacis (Pass.) Sprague & A. G. Johnson, Kilgore, Nebr. Buffalo, Lemmon, Wewela, S. Dak.; Sundance, Wyo.

STIPA LETTERMANII Vasey

Stagonospora simplicior var. nov. Sprague, Skyway, Colo.

STIPA SIBIRICA (L.) Lam.

Pseudomonas coronafaciens var. atropurpureum (Reddy & Godkin) Stapp, Mandan, N. Dak.
Rhizoctonia solani Kuehn, Mandan, N. Dak.

STIPA SPARTEA Trin.

Rhizoctonia solani Kuehn, Minnechaduza Creek, Nebr.

STIPA VIRIDULA Trin.

Pseudomonas coronafaciens var. atropurpureum (Reddy & Godkin) Stapp, Yates, Mont.; Mandan, N. Dak.; Aberdeen, Camp Crook, S. Dak.
Rhizoctonia solani Kuehn, Park River, N. Dak.; Keldron, S. Dak.

TRISETUM CANESCENS Buckl.

Claviceps purpurea (Fr.) Tul., S. F. Clearwater R., Ida.
Septoria calamagrostidis (Lib.) Sacc., S. F. Clearwater R., Ida.

TRITICUM DICOCCUM Schrank

Septoria avenae f. sp. triticea T. Johnson, Mandan, N. Dak.

TRITICUM DURUM Desf.

Septoria avenae f. sp. triticea T. Johnson, Mandan, N. Dak.

WASHINGTON AGRICULTURAL EXPERIMENT STATION AND THE DIVISION OF FORAGE CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE

WHEAT MOSAIC IN WESTERN KANSAS

[NOTE: An article on this outbreak of wheat mosaic has been promised for the REPORTER but is not yet ready. In the meantime, the following press release dated May 24 is quoted, for the record while the epidemic is still news.]

The following joint statement regarding wheat mosaic was issued today by the Bureau of Plant Industry, Soils, and Agricultural Engineering of the U. S. Department of Agriculture and the Kansas Agricultural Experiment Station.

Plant pathologists have this spring noted infestations of mosaic in varying amounts in Kansas wheat fields. The disease is most prevalent in the western third of the State, but has also been noted in a few locations elsewhere.

Wheat mosaic is not a new disease. Scientists noted it in Kansas and Nebraska as early as 1922. Damaging outbreaks occurred in Rawlins and Sheridan Counties in Kansas in 1930, in Saline and Dickinson Counties in 1932, and scattering instances in Northwest Kansas in 1948. Other than that there has been no seriously damaging outbreak until this year.

No official estimate of damage has been made by the U. S. D. A. or the Kansas Agricultural Experiment Station. Reliable estimates on this year's damage cannot be made until the extent of disease infestation on farms has been determined. Such observations as have been made indicate that total damage is very much less than has been estimated in press reports. Once the disease has hit there is no known control for it. The extent of loss from this year's infestation will depend somewhat on weather conditions between now and harvest, but it is probably already too late in the season for much recovery in affected fields. Spread of the disease between now and harvesttime will be insignificant. Experiments in the infested areas in Kansas in the 1930's indicated that infested fields do not remain contaminated from one season to the next.

Wheat mosaics are caused by several distinct viruses, some of which are soil-borne. However, none of the western wheat mosaic viruses have been found to over-season in the soil. Circumstantial evidence indicates the yellow streak mosaic virus may be the chief cause of this year's trouble and that some insect may bring the virus to the young plants in the fall and early spring periods.

Yellow wheat mosaic makes the leaves of the plant striped, mottled, and spotted. It can occur in parts of fields or over entire fields and is capable of stunting plants to the extent of causing a total crop failure. Symptoms first appear in early spring, but in some cases

could be confused with the symptoms of a nitrogen-deficient soil.

Soil samples and plant materials are being collected for detailed laboratory examination. There is a possibility that some of the trouble may be caused by root-rotting organisms and by direct insect damage.

Tests in the Kansas test plots in 1948 indicate that mosaic-resistant varieties of wheat may be developed. The spasmodic appearance of the diseases and the lack of facilities with controlled conditions have retarded progress of study of the diseases and the breeding of resistant varieties.

U. S. BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AND KANSAS AGRICULTURAL EXPERIMENT STATION, PRESS AND RADIO RELEASE, May 24, 1948

EFFECT OF SCLEROTIAL DEVELOPMENT ON INCIDENCE OF AZALEA FLOWER BLIGHT

A. G. Plakidas

In a brief note[1] the writer reported the failure of Ovulinia azaleae to form sclerotia in the Baton Rouge area in 1948 with the following speculative statement:

"It will be of interest to note what effect the failure of sclerotial development will have on the incidence of the disease next year."

This year's observations show that the lack of a heavy initial inoculum load (ascospores) had a very pronounced effect on the incidence of blight.

Early winter was mild. A few blossoms began to open by the end of January. Then, on the last day of January we had a freeze with ice, snow, and sleet. The few open blossoms were killed and further development was retarded for a period of about two weeks. New blossoms began to appear around February 13, and about 50 percent of the blossoms had opened by February 20. Weather conditions during February and March were very favorable for infection, with cool weather, frequent rains, cloudy to partly cloudy days, and foggy nights. There were only two completely sunny days in February.

On February 23, the plant pathology class of 23 students and the instructor examined a large number of azaleas on the Louisiana

1 Plakidas, A. G. Absence of Ovulinia azaleae sclerotia on blighted azalea flowers. Plant Dis. Rept. 32:320. July 15, 1948.

State University campus, searching for infected blossoms and for apothecia. No apothecia were found. However, a few blossoms on two small twigs on separate bushes were found infected. In both cases, it was evident that the infection originated from ascospores because it was localized close to the ground. It is known, of course, that sclerotia will remain viable for more than one year. But it was evident that very few apothecia had developed and only a trace of infection had occurred during the first ten days of flowering.

By February 27, there was localized infection on many bushes, but no general blighting. By March 7, some plants were badly blighted, but the majority of the bushes were covered with healthy flowers. It was strikingly apparent that infection was not spreading, or, at least, not spreading rapidly, from plant to plant. It was a common sight to see plants entirely free of disease only a few yards away from badly blighted ones. The reason for this abnormal behavior was probably this: a limited number of apothecia developed from two-year old sclerotia under a few plants. These started localized infections which spread by splashing rains to other blossoms in the immediate vicinity. Because of inclement weather conditions, insects (particularly bees) were not active, and the infection did not spread to other plants.

March 5, 7, and 8 were clear sunny days and bees were very active. Following heavy and continuous rains on March 13 and 14, the blight became general and severe. Very few healthy blossoms remained after that, but the azalea blooming season was nearing its end anyway.

To summarize, there were about ten days (February 13 to 23) in which there was practically no blight, about two weeks in which blight was localized on a few plants but there was no general spread, a short period (March 7 to 13) in which infection spread rapidly, and this was followed by sudden collapse following the heavy rains of March 13 to 14. The general opinion was that this was the longest and best flowering year for azaleas in the Baton Rouge area since the advent of the disease.

This raises the question of the practicability of using some chemical as a ground eradicant spray that will inhibit apothecial formation. With the large array of organic fungicides available, it would probably not be difficult to find an effective one. A project of this sort presupposes community cooperation. It will not help to apply eradicant sprays to individual plants or individual gardens.

Sclerotia were produced in great profusion this season, and severe blight should be expected next spring if weather conditions are favorable.

LOUISIANA STATE UNIVERSITY, BATON ROUGE

CLADOSPORIUM HEUGELINIANUM ON BUDDLEIA

William W. Diehl

A specimen of Buddleia asiatica Lour. with numerous sooty spots of approximately 2 mm. in diameter on the lower surfaces of the leaves was found by C. L. Lefebvre on March 23, 1949 in a greenhouse in Clarke County, Virginia. The spots are merely masses of conidia and conidiophores of a species of Cladosporium here referred to C. heugelinianum Thuem., and the effect is that of "scab" comparable to the scab of foliage of peach, pecan, apple, etc. Since species and varieties of Buddleia have long been grown in this country and but one other record seems to have been made of it, it is possible that this fungus has either not been noticed or that it has not actually been widely distributed in this country.

The specimen may well be the second finding of the fungus in America, with but one known previous gathering on an unknown species of Buddleia represented by a specimen in the Mycological Collections of the Plant Industry Station, Beltsville, Maryland, made by J. F. Adams at Wilmington, Delaware in 1926 and so identified by G. H. Martin.

The fungus was described by Thuemen (Rev. Myc. 1: p. 11) in 1879 from leaves of B. polystachya, presumably from Eritrea, and appears not to have been recognized since except as recorded above. B. polystachya Fresen. is an East African species of Buddleia. Neither it nor those from South America have become cultigens in the United States. Since no such scab condition of any species of Buddleia is of record, except for those noted above, it is possible that these two records are of hosts have a high degree of susceptibility or that under greenhouse culture susceptibility was more evident.

DIVISION OF MYCOLOGY AND DISEASE SURVEY

RUST IMMUNITY OF CANADIAN BLACK CURRANTS UNDER FIELD CONDITIONS IN CONNECTICUT

G. G. Hahn[1] and Alton Miller[2]

Last year the senior writer reported two hybrid clone selections of Canadian black currants (Ribes ussuriense Jancz. ♂ x R. nigrum L. ♀) that had been derived from seedlings[3], as immune from white-pine blister rust, Cronartium ribicola A. Fisch. These hybrids have been distributed for critical testing to individual horticulturists in five States.[4] Plants have also been distributed to Professor A. J. Riker for pathological study. Under conditions of controlled experimentation with rust inoculum collected in widely separated States, namely, Connecticut, Maine, Virginia and Wisconsin, they have shown freedom from rust infection.

To augment the inoculation tests of 1946-1947, large potted plants of one each of the two hybrid selections, No. O-381 and O-393, now known by the varietal names "Crusader" and "Coronet", respectively, were transplanted into the field, September 1947, for testing with Cronartium ribicola under natural conditions at Litchfield, Connecticut, the following year. The site for the field test selected by the junior writer, who was largely responsible for carrying on the field experiment, possessed a rich soil occurring just inside a protecting roadside stone wall that enclosed a wooded area in which cankered white pines (Pinus strobus L.) were growing. Blister-rust cankers were present within 100 feet of the black currant hybrids. As a "check" on the experiment, two vigorous potted plants of the rust-susceptible wild or pasture gooseberry, Ribes cynosbati L., were planted along with the black currants. The following spring all four test plants developed abundant foliage.

Testing with the hybrids under natural conditions in 1948 was supplemented by artificial dusting May 5 with freshly collected aeciospores of Cronartium ribicola taken from a nearby cankered tree. Since wild ribes in the Litchfield area naturally became infected heavily during the season, this fact clearly indicated that environmental conditions were most suitable for infection. The test plants were observed periodically throughout the spring and summer. Throughout the experiment

---

1 Pathologist, Division of Forest Pathology, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, U. S. Department of Agriculture, in cooperation with Yale University, New Haven, Connecticut.

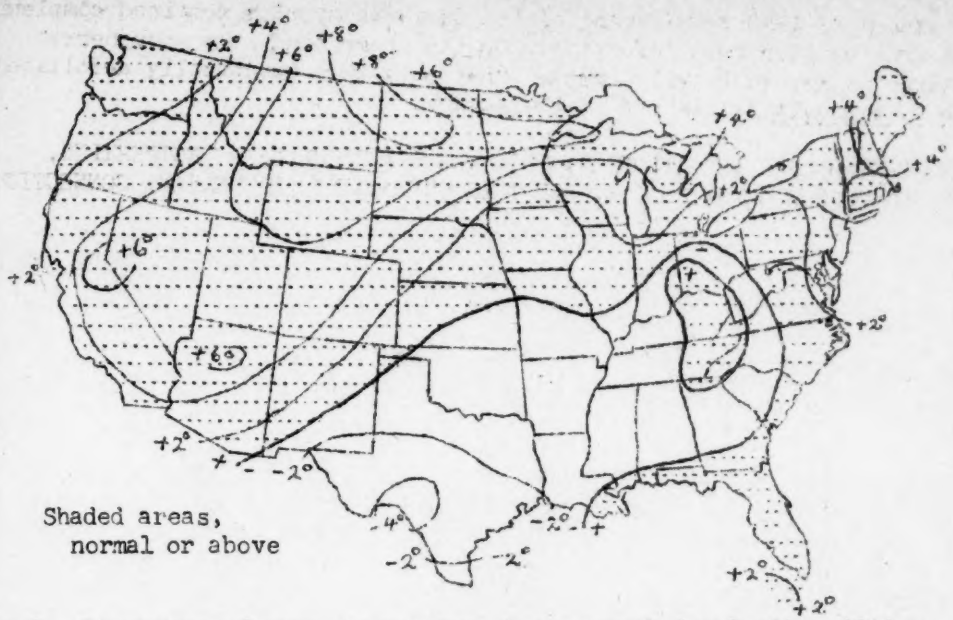
2 Research Assistant, Connecticut Agricultural Experiment Station.

3 Hunter, A. W. S. and M. B. Davis. Breeding rust-resistant black currants. Am. Soc. Hort. Sci. Proc. 42:467-468. 1943.

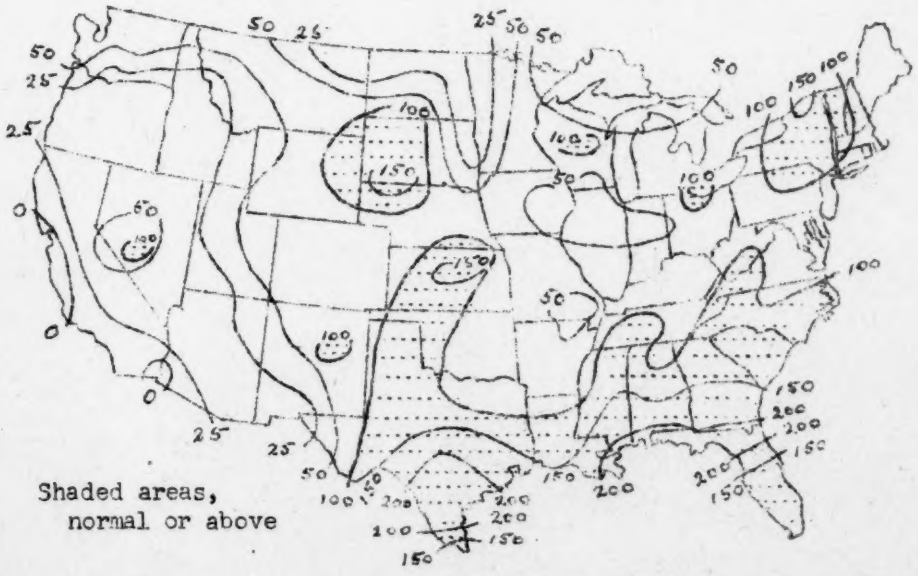
4 Hahn, G. G. Immunity of Canadian black currant selections from blister rust. Phytopath. 38:453-456. 1948.

all stages of leaf development of the Canadian hybrids remained completely free from blister-rust infection. On the other hand, the gooseberry "checks" became so heavily rusted that they were practically defoliated with C. ribicola by the end of the summer.

U. S. BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, DIVISION OF FOREST PATHOLOGY, AND YALE UNIVERSITY, NEW HAVEN, CONNECTICUT



Map I. Departure of mean temperature from normal for April, 1949



Map II. Percentage of normal precipitation for April, 1949

APRIL WEATHER

(From U. S. Department of Commerce, Weather Bureau, Weekly Weather and Crop Bulletin for the week ending May 3, 1949)

LIBRARY
CURRENT SERIAL RECORD
JUN 17 1949
U. S. DEPARTMENT OF AGRICULTURE